Rare case of generalised aggressive periodontitis in the primary dentition

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Abstract: BACKGROUND Generalised aggressive periodontitis (AP) in the prepubescent age is an exceptionally rare disease in the primary dentition of otherwise healthy children. Characteristics of AP are gingival inflammation, deep periodontal pockets, bone loss, tooth mobility and even tooth loss. The most common way of treating this disease is the extraction of all the involved primary teeth. CASE REPORT A 4-year-old girl presented with signs of severe gingival inflammation. Clinical examination revealed deep pockets, increased tooth mobility and bone loss. Microbiological testing revealed the presence of a typical periopathogenic flora consisting of Aggregatibacter actinomycetemcomitans and the typical members of the red complex (Porphyromonas gingivalis, Prevotella intermedia and Treponema denticola). The patient underwent tooth extraction of all primary teeth except the primary canines, followed by thorough root debridement and treatment with systemic antibiotics (amoxicillin plus metronidazole). FOLLOW-UP Regular clinical and microbiological examinations over 4 years showed no signs of recurrence of a periodontitis, even in the erupted permanent teeth. CONCLUSION Early diagnosis and consequent early treatment of aggressive periodontitis can stop the disease and therefore avoid the development of a periodontal disease in the permanent dentition. A close collaboration between specialists of different disciplines is required for a favourable outcome.

DOI: https://doi.org/10.1007/s40368-014-0133-0

Case report: Rare case of generalized aggressive periodontitis in primary teeth

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Introduction

Development of aggressive periodontitis in the primary dentition of otherwise healthy children is - fortunately - an exceptionally rare event. Normally, it affects mostly adults and leads to an advanced loss of periodontal attachment in a short time and may lead - if left untreated - even to tooth loss [Albandar and Tinoco, 2002]. The earliest onset is often circumpubertal [Califano, 2003]. The World Workshop on Periodontal Disease Classification introduced and favored in 1999 the term "aggressive periodontitis" irrespective of the age. Prior to 1999 it was known by other names like "prepubertal periodontitis" and "early onset periodontitis" [Armitage, 1999]. Today AP includes different types of periodontitis that mostly affect people who display systemic health, but a higher familial incidence was reported [American Academy of Periodontology, 2000]. The disease can be found in primary, mixed and permanent dentition and a distinction is made between a localized and a generalized form [Oh et al., 2002]. Both types are frequently associated with the periodontal pathogen Aggregatibacter actinmycetemcomitans (Aa), formerly known as Actinobacillus actinmycetemcomitans [Armitage, 1999].

Clinical characteristics of aggressive periodontitis are gingival inflammation, deep periodontal pockets, loss of attachment, tooth mobility and tooth loss. The diagnosis is generally made from clinical and radiographic examination based on rapid progression characteristics.

An early detection, direct therapy against the infecting microorganisms and providing an environment for healing that is free of infection are major items for a successful treatment of aggressive periodontitis [Califano, 2003]. In 1991 Bimstein emphasized the importance of an early treatment of periodontal diseases in the primary dentition - if present - because periodontitis in the primary dentition may develop into an advanced periodontal disease in the permanent dentition [Bimstein, 1991; Oh et al., 2002].

This report describes and discusses a case of generalized aggressive periodontitis in a four-year old systemically healthy girl.

Case report

A 4-year old girl was referred by her dentist to the Clinic of Orthodontics and Paediatric Dentistry of the University of Zurich due to signs of severe gingival inflammation, deep pockets, increased tooth mobility and bone loss. She had not received any periodontal

therapy before. The parents of the patient observed first signs of gingival inflammation, i.e. bleeding, for about one year.

The clinical oral examination revealed an almost complete primary dentition: Tooth 71 was exfoliated, tooth 31 was already fully erupted (Fig. 1a-c). Oral hygiene was poor and plaque deposits were present around all teeth with severe gingival inflammation. Periodontal probing depths were increased at all teeth. Increased tooth mobility was found in the maxillary incisors. Radiographic examination showed moderate horizontal bone loss of the primary first and second molars. Severe horizontal bone loss was seen around the primary incisors and canines. Horizontal bone loss was also apparent on the permanent incisor 31 (Fig. 2a-f) (Fig 3). Carious lesions could be found on the occlusal surface of the primary mandibular second molars. The medical history of the patient appeared non-contributory.

A microbiological test was performed [The IAI Pado Test 4.5®, Institut für Angewandte Immunologie, Zuchwil, Switzerland] to detect relevant microorganisms typical for advanced periodontitis. For this purpose, subgingival plaque samples were collected at three sites (primary mandibular right first molar, maxillary central incisor, mandibular central incisor) using sterile paper points (Fig. 4). The analysis showed the presence of all four pathogens including Aa, P. gingivalis, P. intermedia and T. denticola (Table 1).

The clinical situation was diagnosed as generalized aggressive periodontitis. Both parents were also screened for periodontal disease and were clinically diagnosed with severe generalized chronic periodontitis and referred for systematic periodontal therapy.

The treatment goal was to avoid the further development and spreading of the severe periodontal disease into the permanent dentition. Therefore, after an interdisciplinary decision-making process, it was decided to extract all primary teeth on the basis of severe periodontal affection (55, 54, 52, 51, 61, 62, 64, 65, 75, 74, 72, 81, 82, 84 and 85) except the canines. The latter and the permanent incisor, however, were maintained to allow for a vertical stabilization on one hand and because they were not severely periodontally compromised yet and treatable. However, a thorough scaling and root planing of these remaining teeth was performed. Due to the age and the cooperation, the treatment was done under general anaesthesia at the Children's Hospital in Zurich. After surgery, the patient was treated with systemic antibiotics for 7 days with 3 daily prescriptions of 350 mg amoxicillin and 3 x 100 mg metronidazole, respectively. There were no reports about any side effects of the antibiotics and parents supervised the intake of medication over the whole

period. In addition, the parents were advised to apply a 0.2% chlorhexidine gel twice a day after tooth brushing for 7 days.

One week after surgery the patient was free of pain. Wound healing proceeded without any complications. Intraoral hygiene was good, nevertheless the parents were re-instructed to brush the girl's teeth at least twice a day.

Follow-up

The patient could be followed-up for four years. The first two years after surgery, recalls were arranged every 6 months and oral hygiene reinstructions and a professional cleaning were made. The oral hygiene was adequate. Clinical and radiographic examination revealed no signs of a periodontal disease at all recall visits. The first molars and the permanent incisors erupted uneventfully and showed no signs of periodontal damage (Fig. 5). Probing depths were below 4 mm without any signs of bleeding (Fig. 6). Another microbiological test four years after surgery detected no periodontal pathogens (Table 1).

Discussion

Severe destruction of periodontal tissue in young children is mostly associated with systemic diseases such as Papillon-Lefèvre syndrome, hypophosphatasia, neutropenia or histiocytosis X [Shaw and Glenwright, 1988; Watanabe et al., 1993; Ishikawa et al., 1994; Kamma et al., 1998; Califano, 2003]. Only a few reports of aggressive periodontitis cases are available in the literature affecting the primary dentition without evidence of an underlying systemic disease [Suzuki et al., 2003; Portaro et al., 2008].

Some studies report that aggressive periodontitis is related with the existence of Aa [Tomita et al., 2013]. The analysis of the subgingival plaque in this case also revealed a massive infection with Aa. Furthermore, the bacterial test showed that the patient also harbored significant amounts of red complex bacteria, i.e. P. gingivalis, P. intermedia and T. denticola. These four pathogens are considered as key pathogenic marker species for advanced periodontitis and are therefore frequently found subgingivally in diseased sites with deep pockets [Holt and Ebersole, 2005]. The question remains, where the young patient acquired these bacteria. Okada and co-workers showed that periodontal bacteria are likely to be

transmitted from parents to children [Okada et al., 2004]. Since both parents of the patient were also positively tested for Aa in the subgingival samples, a vertical infection route may be the most likely explanation. However, relatives may also have an increased risk for periodontitis and share the same bacteria [Dogan et al., 2008].

For a successful treatment outcome, responsible pathogens should preferably be eliminated [Darby and Curtis, 2001]. Several strategies and treatment options can be identified in the literature [Suzuki et al., 2003; Hilgers et al., 2004; Portaro et al., 2008; Cunha et al., 2012]. Clearly, each treatment plan depends on the individual case, based on an adequate examination and diagnosis. In addition, disease progression, bacterial spectrum, the compliance of the patient as well as the prospective peadiatric and orthodontic treatment plan must be taken into careful consideration. Critical for the treatment of children is always the willingness of the whole family to undergo a synoptic therapy. It this case, the treatment was only possible under general anaesthesia. But therapy is also possible under local anaesthesia only, as shown in another case [Cunha et al., 2012].

There is a strong belief, that aggressive periodontitis in the primary teeth is an inevitable precursor to periodontitis in the permanent dentition. Therefore, it was also suggested to extract all primary teeth to prevent the spread of the pathogens to the erupting permanent dentition [Sjodin et al., 1993; Hilgers et al., 2004]. In this context, however, it is noteworthy to point out, that studies showed, that periodontal pathogens may persist even for a long period of time in the oral cavity of edentulous subjects with a history of periodontitis, even in the absence of other hard subgingival surfaces in the mouth and full-mouth extraction [Quirynen and van Assche, 2011]. In addition, the extraction of all primary teeth may cause difficulties in the esthetical and functional situation. In the present case, the canines and the permanent incisors were therefore maintained. There was never a request from patient's or parent's site for prosthetic rehabilitation.

Conclusion

This case report demonstrated that an early diagnosis and consequential early treatment of aggressive periodontitis can stop the disease and therefore avoid the development of a periodontal disease in the permanent dentition. Microbiological testing can help to monitor and detect the responsible microorganisms and to perform targeted treatment measures.

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Figure 1 a-c: (a) Frontal view. **(b)** Maxillary and **(c)** mandibulary aspect before treatment

Fig. 2a-f Radiographic situation before therapy

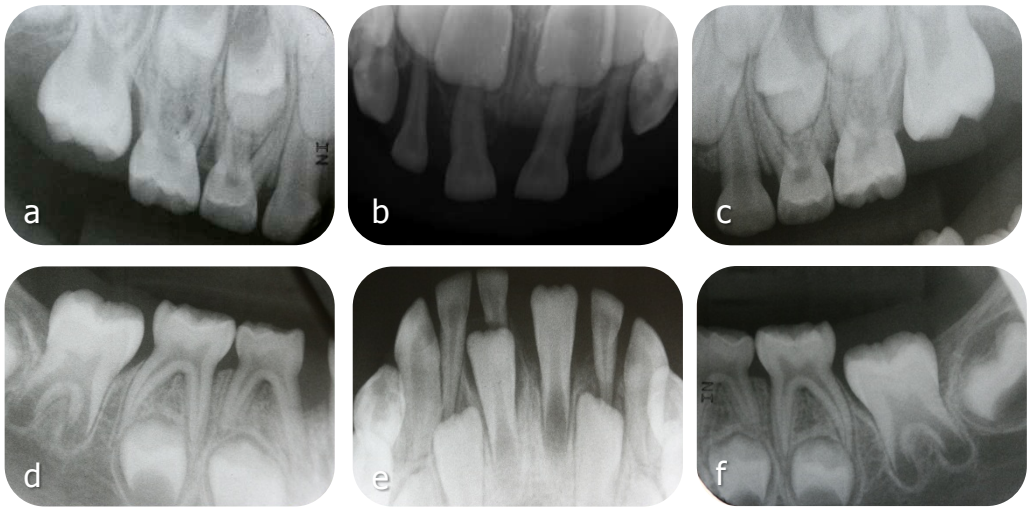

Fig. 3 Probing depth index before treatment

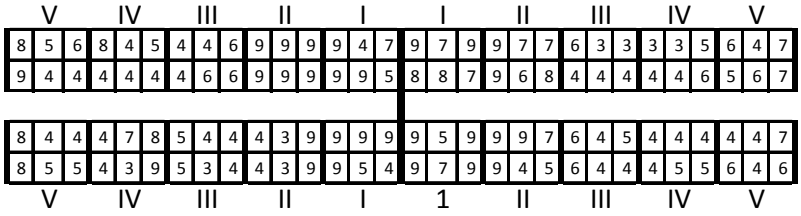

Fig. 4 Subgingival plaque samples were collected at three sites

Fig. 5a-f Radiographic situation one year after therapy.

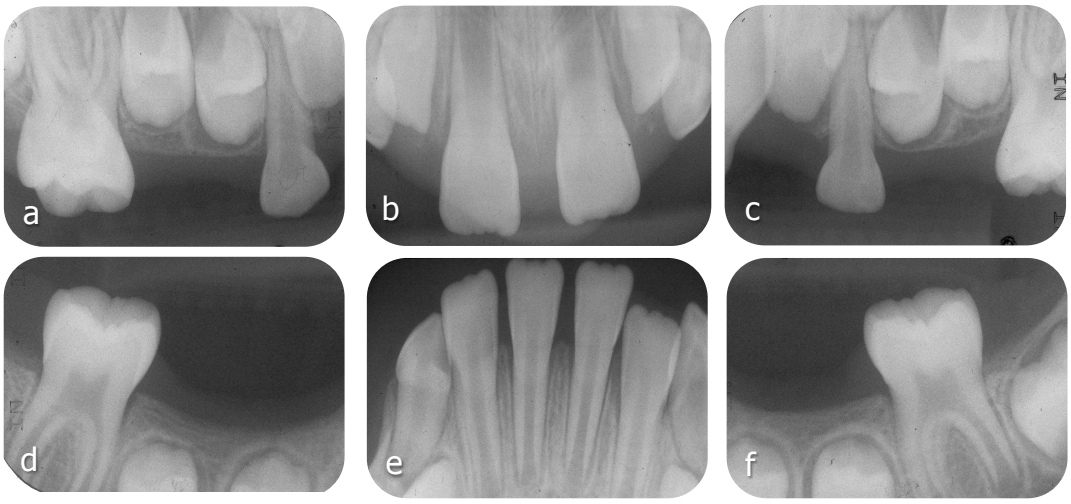

Fig. 6 Probing depth index four years after treatment

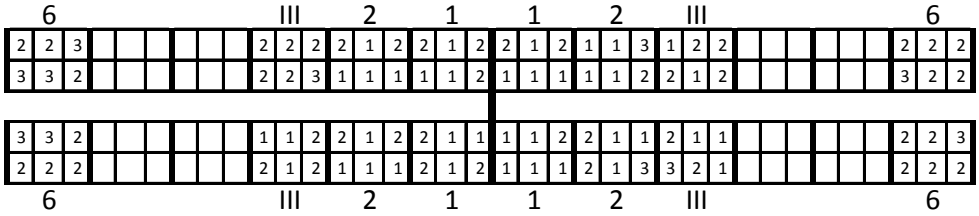

Table 1. Results of microbiological testing for periodontal pathogens before and four years after treatment.

	Aa (n)	Bf (n)	Pg (n)	Td (n)	TBL	TML
Baseline						
Upper front	1.971	0.34	0.17	0.25	78.77	3%
Lower front	0.474	3.15	1.16	4.83	83.51	12 %
4 years						
Teeth (pooled)	0	0.27	0.17	0.45	4.07	21.80%
Tongue	0	0.48	0.3	3.22	8.98	44.60%

Aa = Aggregatibacter actinmycetemcomitans. Bf = Bacteroides forsythus. Pg = Porphyromonas gingivalis. Td = Treponema denticola. TBL = Total Bacterial Load; TML = Total Marker Load. n = Number of bacterias in million.